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ALCOHOL ALERT

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Alcohol and Cognition

Research shows that alcohol adversely affects the brain. When health professionals encounter patients who are having cognitive difficulties, such as impaired memory or reasoning ability, alcohol use may be the cause of the problem. When treating patients who have abused alcohol, it may be of value to attempt to identify the level of any impairment and to modify the treatment accordingly.

Some researchers have investigated whether or not there is measurable alcohol-related cognitive impairment among nonalcoholic social drinkers. Their findings suggest a dose-response relationship between alcohol consumption and diminished scores on certain neuropsychological tests (e.g., Parker & Noble 1977; Parker et al. 1983). Statistically significant decreases in test performance have been found for people whose self-reported alcohol consumption was in the range of what was considered social drinking. This is not to say these people were clinically impaired, only that they exhibited certain performance deficits that correlated with alcohol consumption. It is important to note that similar correlations from other studies have not been found to be consistently significant. For example, the results of one general population study (Bergman et al. 1983) showed no correlation between self-reported alcohol consumption and neuropsychological test scores; other findings (Emmerson et al. 1988) failed to show a simple dose-response relationship. In a recent review of such studies, Parsons (1986) concluded that data on the relationship of cognitive impairment to amount of alcohol consumed by social drinkers are inconclusive.

Alcoholics in treatment present a different picture. Although most alcoholics entering treatment do not have decreased overall intelligence scores, approximately 45 to 70 percent of these patients have specific deficits in problem solving, abstract thinking, concept shifting, psychomotor performance, and difficult memory tasks (Parsons & Leber 1981; Eckardt & Martin 1986; Tabakoff & Petersen 1988). Such deficits usually are not apparent without neuropsychological testing. In addition, structural changes in the brains of alcoholics have been reported (Ron 1979; Wilkinson 1987), as well as reduced cerebral blood flow (Ishikawa et al. 1986) and altered electrical activity (Porjesz & Begleiter 1981), but there is not yet any clear evidence implicating these changes as the cause of observed cognitive deficits.

For the most severe alcoholics, serious organic cerebral impairment is a common complication, occurring in about 10 percent of patients (Horvath 1975). The diverse signs of severe brain dysfunction that persist after cessation of alcohol consumption have been conceptualized in terms of two organic mental disorders: alcohol amnestic disorder (memory disorder) and dementia associated with alcoholism (Lishman 1981; American Psychiatric Association 1987). Recently however, it has been recognized that these two disorders are not mutually exclusive

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Inconclusive data on social drinking and cognitive impairment

Chronic heavy drinking definitely affects cognitive ability

A Commentary by NIAAA Director Enoch Gordis, M.D. 3

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and that some features of each often coexist in the same patient (Martin & Eckardt 1985). Alcohol amnestic disorder, commonly called Korsakoff's psychosis or Wernicke-Korsakoff syndrome, is characterized by short-term memory impairments and behavioral changes that occur without clouding of consciousness or general loss of intellectual abilities. Dementia associated with alcoholism consists of global loss of intellectual abilities with an impairment in memory function, together with disturbance(s) of abstract thinking, judgment, other higher cortical functions, or personality change without a clouding of consciousness. It has been suggested that subcortical lesions due to nutritional (thiamine) deficiency are characteristic of Korsakoff's, whereas alcoholic dementia is associated more with cortical changes (Victor & Laureno 1978). There is some evidence that a genetic abnormality may predispose some people to Korsakoff's in the presence of excessive alcohol use and malnutrition (Blass & Gibson 1977; Mukherjee et al. 1987).

Impairment can be severe

Tarter and Edwards (1986) summarize evidence suggesting that neuropsychological impairment in alcoholics may occur for a number of reasons. The toxic effects of alcohol on the brain may cause impairment directly. In addition, some alcoholics may exhibit impairment as an indirect result of alcohol abuse, e.g., they may have experienced a craniocerebral trauma, they may be eating poorly and suffering nutritional deficits (such as thiamine or niacin deficiencies), or they may have cognitive impairments associated with liver disease.

Some alcoholics may have been cognitively impaired before they began drinking. There is some evidence that persons in groups considered to be at risk for alcoholism (e.g., children of alcoholics) are less adept at certain learning tests and visual-spatial integration than are persons in groups not deemed at risk for alcoholism; this area of research is still under active investigation.

Deficits from aging and alcoholism differ

Some researchers have observed that cognitive deficits in some alcoholics resemble those seen in normal elderly persons, leading to speculation that alcohol's effect on cognition may be explained as premature aging (Tarter & Edwards 1986). However, it is more likely that such deficits are independent of any deficits associated with normal aging (Grant et al. 1984; Cutting 1988).

Is cognitive impairment reversible?

Laying aside issues of etiology, evidence indicates that some cognitive impairment in alcoholics is reversible. Researchers (Albert et al. 1982; Grant et al. 1984; Goldman 1986, 1987) report apparent "spontaneous" recovery of cognitive function (recovery seen after the passage of time with no active intervention) among abstinent alcoholics, a result that may be due solely to the absence of alcohol but that also may be due in part to other changes, such as better nutrition and opportunities for social interaction provided in an alcohol treatment setting. There is some evidence that cognitive training and practice experience (remedial mental exercises) can facilitate recovery from impairment (Godfrey et al. 1985; Goldman 1986, 1987).

Treatment can be affected

Because even with prolonged abstinence many alcoholic patients with chronic organic mental disorders may exhibit only modest clinical improvement in brain functioning, there is a need for pharmacological interventions to complement behavioral methods. Recent findings that pharmacological intervention may be useful in restoring some cognitive ability (McEntee & Mair 1980) are encouraging.

Although degree of cognitive impairment may not be a clinically significant predictor of post-treatment alcohol consumption (Donovan et al. 1987; Eckardt et al. 1988), identifying cognitive impairment may have implications for successfully treating some patients. Particularly in the first weeks of abstinence during treatment, cognitive impairments may make it difficult for some alcoholics to benefit from the educational and skill development sessions that are important components of many treatment programs (McCrady & Smith 1986; McCrady 1987). For example, Becker and Jaffe (1984) reported that alcoholics who were tested soon

after beginning abstinence were unable to recall treatment-related information presented in a film that was part of the regular treatment program. An implication of such findings is that information presented to alcoholics during the period of impairment in the early weeks of abstinence should be repeated at later stages in the treatment program. Alternatively, presentation of treatment-related information should be delayed until tests indicate some improvement in cognitive function.

Tailor treatment to patients' cognitive abilities

Alcohol and Cognition – A Commentary by NIAAA Director Enoch Gordis, M.D.

Awareness of alcohol's effects on cognition can help general health care providers identify alcoholics and refer them to appropriate treatment. Awareness also can assist the efforts of alcoholism treatment personnel to maximize the potential benefit of treatment for their patients.

In general health care situations, practitioners should use standard alcoholism assessment instruments to determine the extent of alcohol use by patients who show signs of cognitive dysfunction. Patients in whom alcohol use is identified as either a primary or a contributing cause of cognitive dysfunction must be referred to alcoholism treatment. Evidence suggests that some cognitive impairment in alcoholics is reversible. Moreover, cognitive deterioration will worsen with continued drinking.

In addition, alcoholism treatment personnel should know that alcohol-induced cognitive impairment may make it difficult during the first weeks of abstinence for some of their patients to benefit from exposure to the full range of treatment services. Although it may not be possible or necessary for treatment programs to administer extensive neuropsychiatric tests to all patients, a simple test of the patient's ability to benefit from the didactic elements of alcoholism treatment can be made. One test might be to determine what a patient remembers from an initial counseling session.

If there is evidence of cognitive deficiency, patients must be allowed time to recover adequate cognitive function so that, at a minimum, information provided during treatment can be retained. However, other important elements of rehabilitation, such as improving patient nutritional status and exposure to physical exercise and resocialization activities, can be undertaken immediately. As cognitive function improves, patients can begin to participate in such treatment components as individual and group therapy, educational programs, and introduction to Alcoholics Anonymous, with a better chance for understanding—and perhaps for acting on—the information provided.

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